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Social Inequalities, Work, and Chronic Disease: Pathways Linking Sociological with Biomedical Research

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ABSTRACT

An important contribution of new knowledge linking sociology with medicine concerns the development of chronic diseases within societies. For a majority of chronic diseases, a social gradient of their distribution has been documented, leaving population groups with lower socioeconomic position at higher risk. While explanations of indirect effects of these social inequalities point to factors such as unhealthy behaviours and delayed or lacking medical care, direct effects explore the pathways from exposure to disadvantageous social conditions to chronic disease development via enhanced stress-related neuronal, neuroendocrine and immune processes. This contribution discusses current evidence on these pathways, based on epidemiologic and naturalistic, quasi-experimental study designs. A robust base of knowledge demonstrates associations of adverse stress-related work and employment conditions with elevated risks of two highly prevalent chronic disorders, ischaemic heart disease and depression. These conditions are more prevalent among lower socioeconomic groups, and they can contribute to the explanation of the social gradient. Importantly, findings point to new preventive strategies, targeting disadvantageous socio-environmental contexts and vulnerable population groups.

Keywords: Health inequalities – ischaemic heart disease – depression - psychosocial work environment- demand-control model – effort-reward imbalance model.

Introduction

In terms of scientific disciplines, medicine and sociology are two very unequal partners as they sharply differ in societal recognition and impact, professional dominance, seniority, and scientific standing. Moreover, their production of scientific knowledge follows two different paradigms, leaving little room for transdisciplinary exchange and collaboration. While the biomedical paradigm analyses phenomena of health and disease in the human organism using experimental evidence derived from basic sciences, including biochemistry, physiology, anatomy and cell biology, a sociological paradigm focuses on the description and explanation of societal structures and processes, and their impact on people's behaviours. Nevertheless, during the second half of the last century, a more complex biopsychosocial model of human health and disease emerged within medicine, and it rapidly expanded from psychiatry and psychosomatic medicine to behavioural and social sciences.[1] This model enabled the inclusion of pioneering sociological knowledge on social determinants of health into a vision of health and disease emerging from permanent exchange between the environment and the individual person. Equally so, it integrated substantial psychological research on personal coping characteristics that moderate effects on health, often in combination with genetic dispositions.[2] Within this new frame of a biopsychosocial model of health and disease, social and behavioural sciences started to explore new associations of their explanatory concepts with biomedical phenomena, most often physical and mental health outcomes. First innovative investigations exploring social determinants of health originated during the post- World War Two period in the United States of America. Examples were an analysis of the health impact of rapid industrialisation in a rural region[3], a study on associations of forced migration with essential hypertension,[4] or an analysis of the effect of occupational instability and mobility on incident coronary heart disease.[5] Yet, these single studies were conducted by epidemiologists and physicians rather than sociologists, often short of theoretical foundation and methodological sophistication.

Different from these investigations medical sociologist Aaron Antonovsky conducted a review of available empirical evidence on social inequalities in life expectancy and overall mortality.[6] A high burden of disease among socially deprived populations was a major discovery of pioneers of public health research in the mid nineteenth century in France, England, and Germany, and it was considered a main characteristic of an exploited working class during early industrialization.[7] However, Antonovsky's results revealed a social gradient of mortality in American population groups around mid-twentieth century. This means that the mortality risk of a population follows a consistent pattern: the higher the socioeconomic position of a population group, the lower its mortality risk. Thus, low life expectancy was not confined to the poorest societal group, as believed for a long time, but its prevalence was reduced by each step a group moved up on the hierarchy of socioeconomic positions. Only a few years after this landmark publication, this surprising observation was confirmed by a large epidemiological study on differential mortality according to level of education and income of the US American population.[8] As this study was based on census data, its validity was not questioned. Furthermore, education data reflected people's long-standing socioeconomic position that acted as a determinant rather than a result of poor health. Again a few years later, probably the first demonstration of a social gradient of mortality in a European country, the United Kingdom, was published by epidemiologist Michael Marmot and his team.[9] This study documented a steep social gradient of coronary heart disease mortality among male British civil servants, using the degree of occupational standing within this organisational hierarchy as main indicator of social inequality.

To date, after several decades of international socio-epidemiological research, the social gradient of morbidity and mortality of a large spectrum of chronic diseases is considered one of the most robust findings of research in epidemiology and public health, and certainly the most important contribution of a sociological concept to the study of chronic disease occurrence. This evidence is particularly convincing in studies originating from Europe. Different from the United States of America, European populations are widely covered by substantial health care provision. High morbidity and mortality therefore cannot be mainly attributed to lack of health care availability. In fact, the range of social differences in mortality in European countries is quite dramatic. For instance, life expectancy among men in the highest income group in the Netherlands is seven years higher than the one of men in the lowest income group.[7] Or in Germany, mean life expectancy among men in the highest income group is 8,6 years higher than among men in the lowest income group. Among women, this difference is 4,4 years.[10] It is therefore important to study social inequalities in chronic disease in more detail. Specifically, what is known about the explanations of this social gradient of morbidity and mortality? Can social factors exert a direct impact on disease development and elevated mortality risk? And if so, are social factors a relevant contextual element within a unified biopsychosocial model of human health and disease?

Study design and explanations of the social gradient of chronic diseases

To tackle these questions, this paper uses a narrative review approach focusing on updated scientific research evidence on one specific explanation of the social gradient of chronic diseases, the role of health-adverse psychosocial working conditions in the onset of two main chronic disease conditions, ischaemic heart disease and depression. As explained below, this selection is justified by three important reasons. First, there exists a clear social gradient of adverse psychosocial working conditions. Second, in current epidemiological research the contribution of these exposures towards explaining elevated risks of the two disorders is particularly well developed. This field of research can therefore instruct explanations of the social gradient of health and disease. Third, complementary results from quasi-experimental and naturalistic studies on psychobiologic pathways underlying the links between stressful work and disease development strengthen the epidemiological evidence,

and they illustrate the significance of a biopsychosocial approach. The paper uses a selective narrative review as systematic reviews on single aspects of the current contribution have already been published (see below) and as any comprehensive systematic review would exceed the current space. Moreover, this contribution offers a unique synthesis of innovative theoretical and empirical knowledge. It starts by describing five explanations of the social gradient of health and disease, focusing then on the role of adverse psychosocial work environments as one of these explanations. Next, the theoretical and methodological bases of research on adverse psychosocial work and its effects on health are explained, and a summary review of main updated results from epidemiologic and naturalistic or quasi-experimental investigations is provided. Finally, some limitations and extensions of this line of research are discussed.

It is crucial to know how social inequalities can be measured. Two approaches were developed in epidemiologic research, an area-based assessment and an assessment based on socioeconomic characteristics of individuals. The former approach defines the material living conditions at the level of neighbourhoods (e.g. at district level), assuming that individuals living in these units share similar conditions of housing and quality of life. In fact, many studies document a linear relationship of level of neighbourhood deprivation with life expectancy.[11] Measuring socioeconomic differences at individual level is a more precise, and more often used approach. Level of income, educational degree, and occupational position are the three main indicators that are applied to define population groups with similar living conditions in socio-epidemiological and public health research. For these measures, international classification systems were developed that enable cross-national comparisons.[7] In research on health inequalities, the three measures differ in their explanatory contribution. As income data may vary across the life span, it is difficult to ensure that they represent a predictor, rather than a consequence of poor health. Occupational position is an important indicator, but it cannot be applied to a large fraction of non-working people. In contrast, educational degree applies to the whole population, and attained in adolescence or early adulthood, it is a rather stable marker throughout life. For these reasons, education is considered the most valid indicator of socioeconomic differences in health in modern societies. Nevertheless, some scientists challenged the notion of education as a purely social indicator, claiming that cognitive ability, an important determinant of educational attainment, in part depends on genetic factors. According to recent study findings, there is a distinct genetic influence on cognitive ability and educational attainment.[12] Yet, this genetic factor cannot invalidate a robust, internationally established base of knowledge pointing to the role of early socialisation and school training in contributing to educational gradients of morbidity and mortality.[13]

The question of how to explain social gradients in health and disease was tackled by a large amount of research initiatives that cannot be summarized here in an appropriate way. Roughly, five types of explanations were proposed. The first type concerns health care. It posits that people with lower income and education face greater difficulties and barriers in access to health services. Lack of care, delayed hospitalization, inadequate treatment, poor adherence to medical advice as well as failure to use preventive services are assumed to increase the burden of disease and the risk of premature mortality among socially more deprived population groups. Several studies support this notion,[14,15] but the contribution of health care towards explaining the social gradient of morbidity and mortality is limited for two reasons. First, as mentioned, many European countries offer extensive health care coverage to their population, thus minimizing unequal access and treatment. Second, whereas the social gradient applies to a wide spectrum of chronic diseases with different amenability to treatment and different severity, including cardiovascular and cerebrovascular diseases, metabolic disorders, respiratory diseases, several cancers, liver disease, depression, dementia as well as functional limitations and accidents, it is unlikely that improved health care could minimize the inequality of occurrence of all these conditions. A second type of explanation focuses on material living conditions. Accordingly, people with fewer protective resources are more frequently exposed to environmental hazards, toxic substances, noise, air pollution, heat, and poor housing conditions, compared to those equipped with more protective resources. Poverty is the main mediator of negative conditions health effects of this exposure.[16]

As poverty is concentrated among population groups at the lower end of a society's social stratification, its contribution towards explaining the entire social gradient of health and disease is again limited, particularly so in countries with extended welfare regimes.[17] Adverse childhood conditions represent a third type of explanations. Systematic reviews document a powerful effect of social deprivation during pregnancy and in infancy on childhood health, often affecting elevated disease susceptibility in adult life.[18,19] This explanation of the social gradient is not only well substantiated by longitudinal life-course studies, but it also offers promising intervention opportunities, given the beneficial effects of health-promoting measures during early life.[20] Still, two complementary explanations are needed to account for unexplained variance in social inequalities in adult health. Health-damaging behaviour is a fourth explanation with two components, a supply side representing legal and illegal markets offering these goods, and a demand side where consumers' motivations, attitudes and overt behaviours play a crucial role. Taken together, this approach is quite powerful as health-adverse behaviours, such as smoking, lack of physical exercise, unhealthy diet, and elevated alcohol consumption, explain the social gradient of mortality to a substantial amount, ranging from twenty up to seventy percent.[21] These behaviours matter most in the development of cardiovascular and metabolic risk factors (hypertension, atherogenic lipids, overweight, diabetes) and of distinct cancers (smoking, alcohol), where their high prevalence among lower socioeconomic groups was shown to be mainly due to a lack of appropriate coping options.[7] The final explanatory approach addresses stressful psychosocial environments in everyday adult life, within families and social networks, and in employment

and working conditions. Although social isolation, social conflicts and poor social support were identified as direct determinants of reduced health and increased mortality,[22] there is less evidence on their socially differential distribution along a gradient. Thus, they may not offer a strong explanatory contribution to health inequalities. With stressful psychosocial employment and working condition, this is different. The lower people's occupational position, the worse their material and psychosocial employment and working conditions.[23,24] A large body of knowledge has defined exposures to stressful psychosocial work environments, has explored its psychobiological pathways to disease development, and has demonstrated associations with the occurrence of highly prevalent chronic diseases. A summary review of this knowledge is given in the next section, with a focus on two chronic diseases, ischaemic (coronary) heart disease and depression.

To conclude, there are good reasons to assume that the social gradient of morbidity and mortality to a considerable extent documents a direct impact of socio-environmental conditions and related behaviours on human health and disease. A large amount of epidemiological research supports this conclusion, offering five complementary explanations. Graded social inequalities in health represent a serious challenge to the health of populations in modern societies, therefore requiring further research on causal roots and implementation of respective knowledge in policies of prevention.[11]

Developing chronic disease: the role of stressful psychosocial work environments

Like any scientific discipline, sociology has the task to define and measure phenomena within its conceptual boundaries, to describe their occurrence and their association with other phenomena of interest, and to explain or predict these associations based on theoretical knowledge. With regard to the current topic, a core descriptive sociological concept, social status, has been selected as starting point of analysis,[25] and this concept has been measured by three complementary indicators, educational degree, level of income, and occupational position. The distribution of social (or socioeconomic) status across society was explored, and associations with health outcomes were studied. As a result, the social gradient of morbidity and mortality was discovered. The next step, the development of explanations, resulted in five complementary approaches. The degree to which sociological theory can contribute to these explanations varies considerably. In case of inequality in health care, concepts derived from economics and from health services research are better suited, and in case of material inequalities, determinants of the physical environment usually provide better predictions than socio-environmental factors. For the three remaining explanations, there is more room for applying sociological (and psychological) theories. While there are promising examples with regard to early childhood[26,27] and health-related behaviours,[7] special emphasis is put here on theoretical models derived from sociology related to unequal exposure to stressful psychosocial work environments. This selection is justified by three reasons. First, the theoretical concepts to be discussed allow a direct application to the analysis of health inequalities as their distribution follows a social gradient. Second, the key terms of these models offer a transdisciplinary link from sociology to biology, as they refer to core notions of psychobiological stress theory. It is important to identify an explanatory model with direct pathways from the social to the biological reality, thus transducing the experience of a stressful psychosocial environment via sensory information and emotional response to autonomic nervous system activation of the organism. Third, during the last three decades, these models were analysed in many epidemiological investigations, documenting associations with some widely prevalent chronic diseases. As a result, a body of solid empirical evidence on these associations is now available.

What do we mean by the term 'psychosocial work environment'? This umbrella term delineates the non-material factors of job tasks and working conditions that are experienced as psycho-mental and socio-emotional challenges or threats. Rapid decision-making, dealing with conflicting situations or frequent interruptions, handling cognitive demands under work pressure, coping with difficult clients, or performing responsible tasks with uncertain outcome are examples of such challenges and threats. With a major shift from the industrial to the service sector of employment, with ground-breaking technological progress of digitization, automation and artificial intelligence, and with the expansion of economic globalization the world of work and employment underwent substantial changes. Dealing with information and communication, computing and disseminating knowledge, offering services to clients through counselling and treatment, and supervising and managing people are highly prevalent job tasks requiring cognitive, affective and social skills rather than physical efforts. As these psychosocial work characteristics became more prominent, occupational health research was faced with the challenge to define those aspects within the complexity and diversity of modern job arrangements that are essential for health and well-being. In order to define these critical elements a theoretical model is needed. A theoretical model selectively identifies a set of elements assumed to be of crucial relevance for health, and these elements are delineated at a high level of abstraction. As generalized notions, these elements can be applied to a wide range of different occupations and professions. Importantly, a theoretical model defines specific interactions between these elements, such that they account for the effects on health and well-being. In terms of research hypotheses, these interactions offer explanations or predictions of the relationships between work environments and health outcomes. In psychosocial occupational health research, several such models were proposed and tested (for review[28-30]). However, few concepts only fulfilled the three criteria mentioned above to analyse health inequalities, that is their prevalence according to a social gradient, the integration of sociological and biological concepts in stress theory, and the availability of a body of knowledge derived from epidemiological investigations. Among these concepts, two theoretical models received particular attention in international research in recent past. Therefore, they were selected for review and discussion.

The first concept, termed 'demand- control (or job strain) model', was developed by American sociologist Robert A. Karasek.[31] Inspired by survey data on occupational characteristics of the American workforce he proposed to focus on two dimensions of job task profiles, the degree of psychological demands (mainly work pressure) put on the working person, and the degree of decision latitude or control available for task execution. Jobs characterized by high demand and low control were assumed to elicit recurrent stressful experience with adverse long-term effects on health and well-being, due to the lack of autonomy at work. Yet, this was not expected from highly demanding jobs with a large degree of decision latitude or control ('active jobs'). In this case, workers were capable to influence their action, to perform successfully and to recover. Two further distinctions are relevant in this model. First, the dimension of control is composed of two different aspects, the worker's influence of performing the task ('decision authority'), and the ability to use own skills ('skill level'). Second, as a further factor, 'social support at work' was later included into this model as a protective resource.[32] However, the majority of empirical studies did not include this factor as their analysis was restricted to the two core dimensions, demand and control. The distribution of this model across occupational categories reveals a strong and consistent social gradient of decision latitude or control, with lower occupational positions documenting less control at work. The association with high demand was less clear as this dimension was highly prevalent among higher occupational positions as well.[33] As a special strength of this model, its core terms offer a transdisciplinary link to psychobiological stress theory. In experimental research, challenging conditions of high demand and low control were shown to simultaneously activate two stress axes within the organism, the sympatho-adrenal medullary and the hypothalamic-pituitary-adrenocortical axis.[34] In the long run, this synergistic activation increases the susceptibility to develop chronic disorders, specifically cardiovascular diseases.[35] With a psychometrically validated questionnaire this model was widely tested in socio-epidemiologic and quasi-experimental research (see below).[36]

As a complementary concept of a stressful psychosocial work environment, the 'effort-reward imbalance model' was developed in own research on medical sociology.[37] With its focus on the employment contract and the centrality of the work role in adult life, it emphasizes the significance of the principle of social reciprocity in costly transactions for health and well-being.[38] An imbalance of high 'cost' (effort) spent with low 'gain' (reward) received in return is assumed to evoke negative emotions of anger and frustration as it violates the core social norm of just transaction. Importantly, three dimensions of reward are distinguished: salary or wage (financial reward), career promotion or job security (status-related reward), and esteem or recognition (socio-emotional reward). Imbalance occurs frequently in modern working life, specifically under conditions of high competition, insecure jobs, and lack of alternative choice in the labour market. The model adds an intrinsic component to the two extrinsic elements, a motivational factor of the working person, defined as 'over-commitment', i.e. an excessive striving and inability to withdraw from work obligations. The model's core hypothesis states that each component, high effort, low reward, and high over-commitment, increases the risk of poor health, but in addition that the imbalance between high effort and low reward matters most. Given the availability of a psychometrically validated short questionnaire,[39] this model has been included in a number of investigations in occupational health research (see below). In several studies, the frequency of this imbalance was shown to follow a social gradient.[40] As was the case with the job strain model, effort-reward imbalance has a strong transdisciplinary link to neuroscience-based stress theory. Sustained reward deficiency activates distinct areas within the brain reward circuits, coding for pain and threat.[41] By activating the organism's core stress axes, it promotes the dysregulation of physiologic systems resulting in allostatic load and elevated risk of stress-related disease development.[42]

Evidence from epidemiologic and quasi-experimental studies

A short summary of findings from prospective occupational cohort studies demonstrates that both theoretical models predict elevated risks of several chronic diseases. Whereas some evidence on musculoskeletal disorders, metabolic syndrome and type 2 diabetes, addictive disorders, and functional limitations including cognitive decline was reported,[43] strongest results were obtained for ischaemic heart disease (IHD; acute myocardial infarction, sudden cardiac death) and for affective disorders (depression). Concerning the demand-control (or job strain) model, a summary estimate of increased relative risk of IHD in the order of thirty to forty percent was observed, based on more than two dozen longitudinal studies.[44] This risk was almost doubled in case of recurrent IHD events, and equally so among men who suffered from cardiometablolic risk factors.[45] Effects of comparable strength were obvious from prospective reports on associations of effort-reward imbalance with IHD, where a risk elevation of forty percent was reported, based on six investigations.[43] Among men exposed simultaneoulsy to both stressful conditions at work, the relative risk was twice as high, compared to the one among men with no stress at work, adjusting for main established cardiovascular risk factors. In this study, women at work did not show a comparable, statistically significant effect.[46]

One cannot claim that these statistical associations represent a causal pathway, without supplementing this evidence by experimental or quasi-experimental results on relationships of stressful psychosocial work with indicators of enhanced autonomic nervous system, inflammation, heart rate, blood pressure, and atherogenic lipids. Importantly, several naturalistic and quasi-experimental investigations demonstrated these links for either work stress model. High blood pressure and hypertension,[47] altered heart rate and heart rate variability,[48] release of catecholamines,[49] atherogenic lipids and fibrinogen,[43] and inflammation[50] are instructive examples. This new information derived from ambulatory monitoring studies in real work settings or from quasi-experimental investigations reveals that IHD is best interpreted as a biopsychosocial disease, where distinct

socioenvironmental (work-related) challenges and threats, in combination with individual coping characteristics, recurrently activate the autonomic nervous system and established cardiovascular risk factors, resulting in long-term functional and structural lesions of the cardiovascular system.[51]

Depression was examined with similar intensity in research focusing on the two work stress models. Several systematic reviews and meta-analyses summarized the findings from multiple studies. A most recent review concluded that the risk of depression was more than twice as high among workers suffering repeatedly from high demand and low control, compared to the risk among those without stressful experience at work. A somewhat lower risk elevation ranging from seventy to ninety percent was reported for the complementary model.[52,53] Of interest, risks of depression following exposure to high effort and low reward are of similar increase among men and women despite the fact that women suffer more frequently from this disease. At least one large study from Finland showed a more than fourfold increased relative risk of disability pension due to depression among those men and women who simultaneously reported job strain and effort-reward imbalance at work.[54] As depression is a risk factor for IHD, and as cardiac patients experience an elevated risk of depression following the onset of clinical events, these findings underscore a considerable clinical relevance. Although depression is a multifactorial disease with a still poorly understood aetiology, a dysregulated hypothalamic-pituitary- adrenocortical axis with resulting alterations in cortisol release and immune competence seems to be involved in the pathogenesis of this disorder. Accordingly, quasi-experimental studies explored relationships of stressful psychosocial work with these biomedical markers. For both theoretical models, associations with distinct immune and inflammatory markers were observed.[55] Moreover, dysregulated diurnal release of cortisol,[56] and elevated hair cortisol,[57] were associated with high effort and low reward at work. More recently, epigenetic markers of accelerated aging - that may increase the susceptibility to develop stress-related chronic disorders - were introduced in occupational health research. Although associations with the two work stress models were not evident, long working hours, an indicator of high demand/high effort as well as high job insecurity and risk of unemployment, a component of low reward,[58] were associated with higher expression of DNA-methylation-based markers of accelerated aging.

Taken together, this selective evidence, though restricted to the two chronic disease conditions of IHD and depression, documents a set of empirical findings that point to a direct impact of distinct socio-environmental conditions, defined as stressful psychosocial work environments, on chronic disease development. This preliminary new knowledge aligns with the more general notion of human health and disease as a biopsychosocial phenomenon, whose analysis requires transdisciplinary research between biomedicine, epidemiology, psychology and sociology. At the current stage, several open questions call for further substantial research investments along these lines.

Limitations and extensions

In this contribution, two chronic disorders with high relevance to public health,[59] ischaemic heart disease and depression, were shown to be partially influenced by distinct factors of the psychosocial work environment. These factors were defined by theoretical models derived from sociology, and they were measured by validated social science methods. Given the independent replication of these associations in studies from different countries, and given a strong study design (prospective observational cohort studies adjusting for confounding factors by multivariable statistical analysis), supplemented by naturalistic and quasi-experimental data on pathways linking exposures with health outcomes, this new sociological evidence represents a significant contribution to medical knowledge. This contribution is even more striking if the larger context of social inequalities in health is considered. The distribution of ischaemic heart disease[9] and of depression[60] across populations follows a social gradient, with higher incidence and prevalence in lower socioeconomic groups. The same holds true for main components of their two partial determinants, demand-control and effort-reward imbalance at work. As a limitation of this paper, the contribution of these latter concepts to the explanation of the social gradient of these disorders has not been analysed in detail. To this end, three mechanisms are relevant, mediation, moderation, and accumulation. In case of mediation, the work stress concepts to a certain extent explain the social gradient of the disease. In several studies, this was shown for both health indicators, although with limited evidence.[53,61] Moderation is present, if the effect size on health attributable to a determinant – in this case the work stress models – increases with each step one moves down on the ladder of socio-economic positions. Among those at the bottom of the societal structure, strongest effects are observed, and this fact is explained by a lack of protective resources and an elevated vulnerability among these population groups. Again, studies have documented a moderation effect of stressful work in interaction with social status on the two health outcomes.[24,62,63] Even in the absence of mediation and moderation, the co-existence of two stressors, lower social status and experience of stressful work, accumulates the burden of coping with adversity, thus aggravating and precipitating disease development.[46] As a further limitation, the exposure assessment of the two work stress models is restricted in two directions. Many studies offer data on only one single assessment. Therefore, the duration of exposure and its change over time cannot be estimated. An important criterion of a causal relationship, the dose-response association between duration of exposure and health outcome, cannot be tested. At the level of measurement, the psychometric scales rely on self-reported information that can be invalidated by reporting bias. This limitation has been addressed by aggregating individual data at work-unit level[64] and by application of a job exposure matrix.[65] Additionally, the exclusive focus on the two models needs to be emphasized. Finally, this selective approach bypasses additional important aspects of stressful work (e.g. mobbing and discrimination) and it does not discuss protective effects, such as personal coping resources that mitigate adversity. [29,30]

Yet, these limitations also point to opportunities of extending this approach in future studies. Advancing explanations of social inequalities in associations of adverse work with these chronic disorders, extending the range of work and employment-related exposures, strengthening the validation of measurements, and developing theory-based intervention studies are examples of such extensions. [66,67] Importantly, applying the core stress-theoretical notions of limited control and disappointed reward in costly social exchange to other life domains, such as family and household work, educational work, caring, informal help or voluntary work, would be essential for the generalisation of these stress-theoretical models. In fact, preliminary research findings support this notion.[68] In summary, despite some limitations, the knowledge reviewed in this paper offers opportunities for practical applications, specifically for improving the quality of psychosocial work environments through worksite health promotion programs, improved leadership, extended screening and monitoring, and implementation of structural measures of organisational and personnel development within companies and enterprises. It is hoped that policy investments will be primarily directed towards disadvantageous, highly vulnerable population groups, thus contributing to a reduction of social inequalities in health.

Conclusion

In conclusion, despite considerable room for extension and modification of research on this topic, the current evidence indicates that persisting social inequalities of chronic diseases, such as ischaemic heart disease and depression, represent a challenge for medicine and public health. Identifying unknown determinants as targets of future preventive efforts is therefore an important scientific task. In this regard, knowledge derived from sociological theory, and applied to stressful psychosocial work environments, proved to be a valuable contribution.

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